WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
L4 and transactivat$	43

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L5

Refine Search | Recall Text | Clear

Search History

DATE: Monday, June 24, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L5	L4 and transactivat$	43	L5
L4	L3 and reporter$	156	L4
L3	(two with hybrid) same (stabl$ or constitutive$)	475	L3
L2	(two with hybrid) same (stabl$ with express)	0	L2
L1	dihybrid same (stabl$ or constitutive$) same (express$ or transfect$)	0	L1

END OF SEARCH HISTORY

WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
L6 and 65	46

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L7

Refine Search | Recall Text | Clear

Search History

DATE: Monday, June 24, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L7	L6 and 65	46	L7
L6	L3 and (stabl$ with transfect$)	68	L6
L5	L4 and transactivat$	43	L5
L4	L3 and reporter$	156	L4
L3	(two with hybrid) same (stabl$ or constitutive$)	475	L3
L2	(two with hybrid) same (stabl$ with express)	0	L2
L1	dihybrid same (stabl$ or constitutive$) same (express$ or transfect$)	0	L1

END OF SEARCH HISTORY

WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
l4 same transact$	5

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L5

Refine Search | Recall Text | Clear

Search History

DATE: Monday, June 24, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L5	l4 same transact$	5	L5
L4	chop	9189	L4
L3	chop$ with transactivat$	0	L3
L2	chop same transactivat$	0	L2
L1	chop same (transactivat$ with domain$)	0	L1

END OF SEARCH HISTORY

WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
chop$ with transactivat$	0

Database:
US Patents Full-Text Database
US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L3

Refine Search

Recall Text | Clear

Search History

DATE: Monday, June 24, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L3	chop$ with transactivat$	0	L3
L2	chop same transactivat$	0	L2
L1	chop same (transactivat$ with domain$)	0	L1

END OF SEARCH HISTORY

```
### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
 ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected


Dialog level 02.05.22D

Last logoff:  18jun02 14:46:19
Logon file405 24jun02 11:55:59
          *** ANNOUNCEMENT ***
                    ***
--Important Notice for Japanese KMKNET Users
KMKNET will be terminated on 5/31/02. Please
switch to DLGNET. Please refer to the G-Search
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libraries.  See HELP LIBRARY for more information.
                    ***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
                    ***
For information about the access to file 43 please see Help News43.
***
NEW FILES RELEASED
***AGROProjects (File 235)
***ARCHIVES OF DERMATOLOGY - SUBSCRIBERS (File 787)
***ARCHIVES OF GENERAL PSYCHIATRY -SUBSCRIBERS (File 794)
***ARCHIVES OF INTERNAL MEDICINE - SUBSCRIBERS(File 795)
***ARCHIVES OF NEUROLOGY - SUBSCRIBERS (File 796)
***ARCHIVES OF OPHTHALMOLOGY - SUBSCRIBERS (File 797)
***ARCHIVES OF OTOLARYNGOLOGY - SUBSCRIBERS(File 798)
***ARCHIVES OF PEDIATRIC & ADOLESCENT MEDICINE-
Subscribers (File 789)
***ARCHIVES OF SURGERY - SUBSCRIBERS (File 800)
***JAMA - Journal of the American Medical Association -
   Subscribers (File 785)
***MIRA (File 81)
***TRADEMARKSCAN-Japan (File 669)
                    ***
UPDATING RESUMED
***Delphes European Business (File 481)
                    ***
RELOADED
***CLAIMS/US PATENTS (Files 340, 341, 942)
***Kompass Western Europe (590)
***D&B - Dun's Market Identifiers  (516)

REMOVED
***Los Angeles Times (File 630)
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***Baton Rouge Advocate (File 382)
***Washington Post (File 146)
***Books in Print (File 470)
***Court Filings (File 793)
***Microcomputer Software Guide Online (File 278)
***Publishers, Distributors & Wholesalers of the U.S. (File 450)
***State Tax Today (File 791)
***Tax Notes Today (File 790)
***Worldwide Tax Daily (File 792)

***New document supplier***
IMED has been changed to INFOTRIE (see HELP OINFOTRI)


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     >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
     >>>    of new databases, price changes, etc.      <<<
                 ****
KWIC is set to 50.
HILIGHT set on as '*'
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.7.8 term=ASCII
                    *** DIALOG HOMEBASE(SM) Main Menu ***

 Information:
  1.  Announcements (new files, reloads, etc.)
  2.  Database, Rates, & Command Descriptions
  3.  Help in Choosing Databases for Your Topic
  4.  Customer Services (telephone assistance, training, seminars, etc.)
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  7.  Data Star(R)

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      /H = Help            /L = Logoff          /NOMENU = Command Mode


Enter an option number to view information or to connect to an online
 service.  Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
?b biotech biochem medicine

       24jun02 11:56:11 User243084 Session D280.1
            $0.00    0.157 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.04  TELNET
     $0.04  Estimated cost this search
     $0.04  Estimated total session cost   0.157 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R)  1969-2002/Jun W3
         (c) 2002 BIOSIS
  File   6:NTIS  1964-2002/Jul W1
         (c) 2002 NTIS, Intl Cpyrght All Rights Res
*File   6: See HELP CODES6 for a short list of the Subject Heading Codes
```

(SC=, SH=) used in NTIS.
File 8:Ei Compendex(R) 1970-2002/Jun W4
(c) 2002 Engineering Info. Inc.
File 34:SciSearch(R) Cited Ref Sci 1990-2002/Jun W4
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File 71:ELSEVIER BIOBASE 1994-2002/Jun W4
(c) 2002 Elsevier Science B.V.
File 73:EMBASE 1974-2002/Jun W3
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see Help News73.
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File 94:JICST-EPlus 1985-2002/May W1
(c)2002 Japan Science and Tech Corp(JST)
***File 94: There is no data missing. UDs have been adjusted to reflect**
the current months data. See Help News94 for details.
File 98:General Sci Abs/Full-Text 1984-2002/May
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File 99:Wilson Appl. Sci & Tech Abs 1983-2002/May
(c) 2002 The HW Wilson Co.
File 135:NewsRx Weekly Reports 1995-2002/Apr W1
(c) 2002 NewsRx
File 143:Biol. & Agric. Index 1983-2002/May
(c) 2002 The HW Wilson Co
File 144:Pascal 1973-2002/Jun W4
(c) 2002 INIST/CNRS
File 155:MEDLINE(R) 1966-2002/Jun W3
***File 155: Daily alerts are now available. This file has**
been reloaded. Accession numbers have changed.
File 172:EMBASE Alert 2002/Jun W4
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Derwent announces file enhancements. Please see HELP NEWS 357.
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***File 358: Updates delayed. Please see HELP NEWS 358 for details.**
File 369:New Scientist 1994-2002/Jun W2
(c) 2002 Reed Business Information Ltd.
File 370:Science 1996-1999/Jul W3
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***File 370: This file is closed (no updates). Use File 47 for more current**
information.
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***File 399: Use is subject to the terms of your user/customer agreement.**
RANK charge added; see HELP RATES 399.
File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
(c) 1998 Inst for Sci Info
File 40:Enviroline(R) 1975-2002/May
File 41:Pollution Abs 1970-2002/Jul
(c) 2002 Cambridge Scientific Abstracts
File 50:CAB Abstracts 1972-2002/May
(c) 2002 CAB International
***File 50: Truncating CC codes is recommended for full retrieval.**
See Help News50 for details.
File 68:Env.Bib. 1972-2002/May
(c) 2002 Internl Academy at Santa Barbara
File 77:Conference Papers Index 1973-2002/May

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File 103:Energy SciTec[redacted]1974-2002/Jun B1
(c) 2002 Contains copyrighted material
***File 103: For access restrictions see Help Restrict.**
File 156:ToxFile 1966-2002/Mar W4
(c) 2002
File 162:CAB HEALTH 1983-2002/May
(c) 2002 CAB INTERNATIONAL
***File 162: Truncating CC codes is recommended for full retrieval.**
See Help News162 for details.
File 305:Analytical Abstracts 1980-2002/Jun W2
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***File 305: Frequency of updates and Alerts changing to weekly.**
See HELP NEWS 305.
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File 48:SPORTDiscus 1962-2002/Jun
(c) 2002 Sport Information Resource Centre
File 91:MANTIS(TM) 1880-2002/Aug
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File 149:TGG Health&Wellness DB(SM) 1976-2002/Jun W3
(c) 2002 The Gale Group
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***File 467: For information about updating status please see Help News467.**

Set	Items	Description
?s chop? (s) transactiv?		
	55955	CHOP?
	83015	TRANSACTIV?
S1	73	CHOP? (S) TRANSACTIV?

?rd
...examined 50 records (50)
...completed examining records

S2	19	RD (unique items)

?show files;ds;t/3,k/all
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File 135:NewsRx Weekly Reports 1995-2002/Apr W1
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File 143:Biol. & Agric. Index 1983-2002/May
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Set Items Description
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S2 19 RD (unique items)
>>>KWIC option is not available in file(s): 41, 77, 399

2/3,K/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

13688188 BIOSIS NO.: 200200317009
The retinoid-inducible gene I: Effect on apoptosis and mitogen-activated kinase signal pathways.
AUTHOR: Huang Shiang-Long; Shyu Rong-Yaun; Yeh Ming-Yang; Jiang Shun-Yuan (a)
AUTHOR ADDRESS: (a)Department of Microbiology and Immunology, National Defense Medical Center, Taipei, 114**Taiwan E-Mail: jsy@ndmctsgh.edu.tw
JOURNAL: Anticancer Research 22 (2A):p799-804 March-April, 2002
MEDIUM: print
ISSN: 0250-7005
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: RIG1-EGFP and RIG1-myc fusion proteins induced cellular apoptosis that was characterized by the presence of apoptotic bodies and in situ DNA breakage. The *transactivation* activities of Elk1, c-Jun and *CHOP* proteins were suppressed by 80, 50 and 88%, respectively, in HtTA cells expressing the RIG1-myc fusion protein for two days. Similarly, the *transactivation* activities of the *CHOP* protein was suppressed in TSGH9201 and HtTA cells transiently expressing RIG1-myc and RIG1-EGFP, respectively. Conclusion: The RIG1 fusion proteins exhibited growth suppressive and...

2/3,K/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13094154 BIOSIS NO.: 200100301303
Involvement of the pro-oncoprotein TLS (translocated in liposarcoma) in nuclear factor-kappaB p65-mediated transcription as a coactivator.
AUTHOR: Uranishi Hiroaki; Tetsuka Toshifumi; Yamashita Mayumi; Asamitsu Kaori; Shimizu Manabu; Itoh Makoto; Okamoto Takashi(a)
AUTHOR ADDRESS: (a)Department of Molecular Genetics, Nagoya City University Medical School, 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya, Aichi, 467-8601: tokamoto@med.nagoya-cu.ac.jp**Japan
JOURNAL: Journal of Biological Chemistry 276 (16):p13395-13401 April 20, 2001
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

...ABSTRACT: by a coimmunoprecipitation experiment followed by Western blot of the cultured cell in vivo. TLS was originally identified as part of a fusion protein with *CHOP* arising from chromosomal translocation in human myxoid liposarcomas. TLS has been shown to be involved in TFIID complex formation and associated with RNA polymerase II. However, the role of TLS in transcriptional regulation has not yet been clearly elucidated. We found that TLS enhanced the NF-kappaB-mediated *transactivation* induced by physiological stimuli such as tumor necrosis factor alpha, interleukin-1beta, and overexpression of NF-kappaB-inducing kinase. TLS augmented NF-kappaB-dependent promoter...

...interferon-beta gene. These results suggest that TLS acts as a coactivator of NF-kappaB and plays a pivotal role in the NF-kappaB-mediated *transactivation*.

2/3,K/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12794529 BIOSIS NO.: 200100001678
Role of C/EBP homologous protein (CHOP-10) in the programmed activation of CCAAT/enhancer-binding protein-beta during adipogenesis.
AUTHOR: Tang Qi-Qun(a); Lane M Daniel
AUTHOR ADDRESS: (a)Department of Biological Chemistry, The Johns Hopkins University School of Medicine, Baltimore, MD, 21209: qqtang@welchink.welch.jhu.edu**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (23):p12446-12450 November 7, 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

...ABSTRACT: binding activity and fails to localize to centromerers until preadipocytes traverse the G1-S checkpoint of mitotic clonal expansion. Evidence is presented that dominant-negative *CHOP*-10 expressed by growth-arrested preadipocytes transiently sequesters C/EBPbeta by heterodimerization. As preadipocytes reach S phase, *CHOP*-10 is down-regulated, apparently releasing C/EBPbeta from inhibitory constraint and allowing *transactivation* of the C/EBPalpha gene. In support of these findings, up-regulation of *CHOP*-10 with the protease inhibitor N-acetyl-Leu-Leu-norleucinal prevents activation of C/EBPbeta, expression of C/EBPalpha, and adipogenesis.

2/3,K/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12623236 BIOSIS NO.: 200000376738
A novel zinc finger gene is fused to EWS in small round cell tumor.
AUTHOR: Mastrangelo Tiziana; Modena Piergiorgio; Tornielli Silvana; Bullrich Florentia; Testi Maria Adele; Mezzelani Alessandra; Radice Paolo ; Azzarelli Alberto; Pilotti Silvana; Croce Carlo M; Pierotti Marco A; Sozzi Gabriella(a)
AUTHOR ADDRESS: (a)Istituto Nazionale Tumori, Via G. Venezian 1, 20133, Milano**Italy
JOURNAL: Oncogene 19 (33):p3799-3804 3 August, 2000
MEDIUM: print
ISSN: 0950-9232
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

...ABSTRACT: 22q12 to five different members of transcription factors namely FLI-1, ERG, ETV1, E1AF and FEV. Different classes of DNA binding proteins, ATF1, WT1 and *CHOP* are fused to EWS generating distinct tumor phenotypes: clear cell sarcoma, desmoplastic small round cell tumor, and myxoid liposarcoma, respectively. We have cloned a novel...
...repressor-like domain at the N-terminus. The rearrangement involves intron 8 of EWS and exon 1 of ZSG creating a chimeric sequence containing the *transactivation* domain of EWS fused to zinc finger domain of ZSG. This product lacks the transcriptional repressor domain at the N-terminus of ZSG. A rearrangement...

2/3,K/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12273559 BIOSIS NO.: 200000027061
CHOP enhancement of gene transcription by interactions with Jun/Fos AP-1 complex proteins.
AUTHOR: Ubeda Mariano; Vallejo Mario; Habener Joel F(a)
AUTHOR ADDRESS: (a)Laboratory of Molecular Endocrinology, Massachusetts General Hospital, 55 Fruit St., WEL320, Boston, MA, 02114-2696**USA
JOURNAL: Molecular and Cellular Biology 19 (11):p7589-7599 Nov., 1999
ISSN: 0270-7306
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: The transcription factor *CHOP* (C/EBP homologous protein 10) is a bZIP protein induced by a variety of stimuli that evoke cellular stress responses and has been shown to arrest cell growth and to promote programmed cell death. *CHOP* cannot form homodimers but forms stable heterodimers with the C/EBP family of activating transcription factors. Although initially characterized as a dominant negative inhibitor of C/EBPs in the activation of gene transcription, *CHOP*-C/EBP can activate certain target genes. Here we show that *CHOP* interacts with members of the immediate-early response, growth-promoting AP-1 transcription factor family, JunD, c-Jun, and c-Fos, to activate promoter elements in the somatostatin, JunD, and collagenase genes. The leucine zipper dimerization domain is required for interactions with AP-1 proteins and *transactivation* of transcription. Analyses by electrophoretic mobility shift assays and by an in vivo mammalian two-hybrid system for protein-protein interactions indicate that *CHOP* interacts with AP-1 proteins inside cells and suggest that it is recruited to the AP-1 complex by a tethering mechanism rather than by direct binding of DNA. Thus, *CHOP* not only is a negative or a positive regulator of C/EBP target genes but also, when tethered to AP-1 factors, can activate AP-1 target genes. These findings establish the existence of a new mechanism by which *CHOP* regulates gene expression when cells are exposed to cellular stress.

2/3,K/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11973935 BIOSIS NO.: 199900227248
Complexes containing activating transcription factor (ATF)/cAMP-responsive-element-binding protein (CREB) interact with the CCAAT/enhancer-binding protein (C/EBP)-ATF composite site to regulate Gadd153 expression during the stress response.
AUTHOR: Fawcett Timothy W; Martindale Jennifer L; Guyton Kathryn Z; Hai Tsonwin; Holbrook Nikki J(a)
AUTHOR ADDRESS: (a)Gene Expression and Aging Section, Laboratory of Biological Chemistry, NIA, National Institutes **USA
JOURNAL: Biochemical Journal 339 (1):p135-141 April 1, 1999
ISSN: 0264-6021
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Gadd153, also known as *chop*, encodes a member of the CCAAT/enhancer-binding protein (C/EBP) transcription factor family and is transcriptionally activated by cellular stress signals. We recently demonstrated...

...expression declined. [redacted] further demonstrated that ATF4 [redacted]tivates, while ATF3 represses, Gadd153 promoter activity through the C/EBP-ATF site. ATF3 also repressed ATF4-mediated *transactivation* and arsenite-induced activation of the Gadd153 promoter. Our results suggest that numerous members of the ATF/CREB family are involved in the cellular stress...

2/3,K/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11314307 BIOSIS NO.: 199800095639
TLS (translocated-in-liposarcoma) is a high-affinity interactor for steroid, thyroid hormone, and retinoid receptors.
AUTHOR: Powers C Andrew(a); Mathur Mukul; Raaka Bruce M; Ron David; Samuels Hebert H
AUTHOR ADDRESS: (a)Div. Mol. Endocrinol., N.Y. Univ. Med. Cent., New York, NY 10016**USA
JOURNAL: Molecular Endocrinology 12 (1):p4-18 Jan., 1998
ISSN: 0888-8809
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: in vivo. TLS was originally discovered as part of a fusion protein arising from a chromosomal translocation causing human myxoid liposarcomas. TLS contains a potent *transactivation* domain whose translocation-induced fusion with a DNA-binding protein (*CHOP*) yields a powerful transforming oncogene and transcription factor. The *transactivation* and RNA-binding properties of TLS and the nature of its interaction with nuclear receptors suggest a novel role in nuclear receptor function.

2/3,K/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11085617 BIOSIS NO.: 199799706762
The hepatitis B virus X protein enhances the DNA binding potential and transcription efficacy of bZip transcription factors.
AUTHOR: Barnabas Sangeeta; Hai Tsonwin; Andrisani Ourania M(a)
AUTHOR ADDRESS: (a)Dep. Basic Med. Sci., Sch. Veterinary Med., Purdue Univ., 1246 Lynn Hall, West Lafayette, IN 479**USA
JOURNAL: Journal of Biological Chemistry 272 (33):p20684-20690 1997
ISSN: 0021-9258
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: O. M. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 3819-3823). Here we examine pX interactions with bZip transcription factors ATF-3, gadd153/*Chop10*, ICER II-gamma, and NF-IL6. We demonstrate direct interactions in vitro between pX and the bZip proteins tested. In contrast MyoD and Gal4-1...

...involvement of pX in gene repression. We conclude that pX is an enhancer of the DNA binding potential of bZip transcription factors, thereby increasing the *transactivation* or repression efficacy of bZip-responsive genes.

2/3,K/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09443292 BIOSIS NO.: 199497451662
Transcriptional activation by TAL1 and FUS-CHOP proteins expressed in acute

malignancies as a result of chromosomal abnormalities.
AUTHOR: Sanchez-Garcia I; Rabbitts T H
AUTHOR ADDRESS: Med. Res. Council Lab., Mol. Biol., Hills Rd., Cambridge CB2 2QH**UK
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 91 (17):p7869-7873 1994
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: ectopically activated in T-cell acute leukemias after chromosomal abnormalities caused by V-D-J recombinase error) (V, variable; D, diversity; J, joining) and FUS-*CHOP* (a liposarcoma tumor-specific fusion protein that is produced as a result of a chromosomal translocation) can function as transcription activators of specific responsive reporter...

...provides evidence that transcriptional activation can be mediated by a gene activated by translocation in T-cell acute leukemias. In the case of the liposarcoma, *transactivation* by the FUS-*CHOP* protein occurs because the FUS transcriptional activation domain is added to the DNA-binding *CHOP* protein normally lacking such activity. Therefore, the association of transcriptional activation and DNA-binding elements is a common consequence in proteins activated or newly created...

2/3,K/10 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06829723 Genuine Article#: ZV238 No. References: 32
Title: Human translocation liposarcoma CCAAT enhancer binding protein (C/EBP) homologous protein (TLS-CHOP) oncoprotein prevents adipocyte differentiation by directly interfering with C/EBP beta function
Author(s): Adelmant G; Gilbert JD; Freytag SO (REPRINT)
Corporate Source: HENRY FORD HLTH SYST,DEPT BIOL MOL, 1 FORD PL, WING 5D/DETROIT//MI/48202 (REPRINT); HENRY FORD HLTH SYST,DEPT BIOL MOL/DETROIT//MI/48202; HENRY FORD HLTH SYST,DEPT RADIAT ONCOL/DETROIT//MI/48202
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1998, V273, N25 (JUN 19), P 15574-15581
ISSN: 0021-9258 Publication date: 19980619
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

Abstract: Human translocation liposarcoma (TLS)-CCAAT/enhancer binding protein (C/EBP) homologous protein (*CHOP*) is a fusion oncoprotein found specifically in a malignant tumor of adipose tissue and results from a t(12;16) translocation that fuses the amino-terminal part of TLS to the entire coding region of *CHOP*. Being that *CHOP* is a member of the C/EBP transcription factor family, proteins that comprise part of the adipocyte differentiation machinery, we examined whether TLS-*CHOP* blocked adipocyte differentiation by directly interfering with C/EBP function, Using a single-step retroviral infection protocol, either wild-type or mutant TLS-*CHOP* were co-expressed along with C/EBP beta in naive NIH3T3 cells, and their ability to inhibit C/EBP beta driven adipogenesis was determined, TLS-*CHOP* was extremely effective at blocking adipocyte differentiation when expressed at a level comparable to that observed in human myxoid liposarcoma. This effect of TLS-*CHOP* required a functional leucine zipper domain and correlated with its ability to heterodimerize with C/EBP beta and inhibit C/EBP beta DNA binding and *transactivation* activity in situ. In contrast, the TLS-*CHOP* basic region was dispensable, making it unlikely that the inhibitory effect of TLS-*CHOP* is attributable to unscheduled gene expression resulting from TLS-*CHOP*'s putative *transactivation*

activity. Another adipogenic transcription factor, PPARgamma 2, was able to rescue TLS-*CHOP*-inhibited cells, indicating that TLS-*CHOP* interferes primarily with C/EBP beta-driven adipogenesis and not with other requisite events of the adipocyte differentiation program. Together, the results demonstrate that TLS-*CHOP* blocks adipocyte differentiation by directly preventing C/EBP beta from binding to and *transactivating* its target genes. Moreover, they provide strong support for the thesis that a blockade to normal differentiation is an important aspect of the cancer process.

2/3,K/11 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04238246 Genuine Article#: RQ624 No. References: 95
Title: THE EMERGING MOLECULAR-GENETICS OF SARCOMA TRANSLOCATIONS
Author(s): LADANYI M
Corporate Source: MEM SLOAN KETTERING CANC CTR,DEPT PATHOL,1275 YORK AVE/NEW YORK//NY/10021; MEM SLOAN KETTERING CANC CTR,DEPT HUMAN GENET/NEW YORK//NY/10021
Journal: DIAGNOSTIC MOLECULAR PATHOLOGY, 1995, V4, N3 (SEP), P162-173
ISSN: 1052-9551
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

...Abstract: consisting in the fusion of a gene with an RNA-binding domain (EWS or TLS) with a transcription factor gene (FLI1, ERG, ETV1, ATF-1, *CHOP*, or WT1). The observation that the different translocation partners of the EWS gene are specifically associated with several distinct types of primitive sarcomas suggests a...

...the target specificity of the transcriptional activation mediated by these chimeric proteins, whereas the partner supplying the N-terminal domain and promoter region determines their *transactivation* potential and expression level. Further analysis of the normal functions and expression patterns of these genes should yield insights into the histogenesis of these different...

2/3,K/12 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03751718 H.W. WILSON RECORD NUMBER: BGSI98001718 (USE FORMAT 7 FOR FULLTEXT)
Consequences of chromosomal abnormalities in tumor development.
Sanchez-Garc a, I
Annual Review of Genetics (Annu Rev Genet) v. 31 ('97) p. 429-53
SPECIAL FEATURES: bibl il ISSN: 0066-4197
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 10415

(USE FORMAT 7 FOR FULLTEXT)

TEXT:
... The promyelocytic leukemia Kruppel-like zinc finger (PLZF) protein normally represses expression of a cyclin A promoter reporter gene, but the PLZF-RARa fusion protein *transactivates* cyclin A expression (19), which is essential for cell cycle progression and growth. *CHOP*, which was fingered as an oncoprotein through its identification as a chromosomal translocation fusion partner of the EWS RNA-binding protein in a myxoid liposarcoma, blocks differentiation of adipocytes (6). Transformation of fibroblasts requires a contribution from the EWS fusion partner, but *CHOP* may act as an oncoprotein by preventing expression of differentiation genes. PML, which was uncovered as a translocation partner of RARa in promyelocytic leukemia (PML...

2/3,K/13 (Item 1 from file: 370)
DIALOG(R)File 370:Science

00500410 (USE 9 FOR FULLTEXT)

Stress-Induced Phosphorylation and Activation of the Transcription Factor CHOP (GADD153) by p38 MAP Kinase

Wang, XiaoZhong; Ron, David
Departments of Medicine and Cell Biology, Skirball Institute of Biomolecular Medicine, and the Kaplan Cancer Center, New York University Medical Center, New York, NY 10016, USA.

Science Vol. 272 5266 pp. 1347
Publication Date: 5-31-1996 (960531) Publication Year: 1996
Document Type: Journal ISSN: 0036-8075
Language: English
Section Heading: Reports
Word Count: 2164

(THIS IS THE FULLTEXT)

Text: *CHOP*, also known as growth arrest and DNA damage-inducible gene 153 (GADD153), is expressed in response to various metabolic stresses in all cells tested (B1) . By forming heterodimers with members of the C/EBP family of transcription factors, *CHOP* influences gene expression as both a dominant-negative regulator of C/EBP binding to one class of DNA targets (B2) and by directing *CHOP*-C/EBP heterodimers to other sequences (B3) . Both modes of action are implicated in the effects of *CHOP* on cellular growth (B4) (B5) and differentiation (B6) . In addition to inducing *CHOP* expression, stress increases the ability of *CHOP* to activate gene expression (B3) . This latter observation prompted us to study the possible stress-induced posttranslational modification of *CHOP*.

...

...In stressed cells, *CHOP* is present in two forms, distinguishable by their migration in SDS-polyacrylamide gel electrophoresis (Fig. 1A) (B7) . Isoelectric focusing revealed the form of *CHOP* with decreased mobility to have a more acidic isoelectric point, suggestive of phosphorylation. To study the possible effects of stress on *CHOP* phosphorylation independently of the stress-induced increase in protein, we used cells constitutively expressing an epitope-tagged form of *CHOP* that is distinguishable in size from the endogenous protein. In vivo labeling with [.sup(32)P]orthophosphate followed by immunoprecipitation revealed two to four times as much phosphorylation of tagged *CHOP* in response to stress with no change in the amount of protein (Fig. 1B). The stress-induced endogenous *CHOP* was also phosphorylated...

...Comparison of the tryptic phosphopeptide maps of the tagged *CHOP* from stressed and unstressed cells showed most of the inducible phosphorylation to occur on two distinct peptides (Fig. 1C). All known *CHOP* proteins contain two adjacent serine residues [amino acids 78 and 81 in the mouse sequence (B2)] in a context that may serve as a substrate for members of the MAP kinase family (Fig. 1E). Members of this family are activated by many of the same insults that induce *CHOP* expression (B8) and may therefore participate in the stress-induced phosphorylation of *CHOP*. Conversion of Ser.sup(78) or Ser.sup(81) to Ala led to the selective loss of inducible phosphorylation of one tryptic peptide (Fig. 1C). The minimal residual phosphorylation of the Ala-substituted peptides may have resulted from the presence of other phosphorylated residues. The mutant *CHOP* proteins exhibited substantially less total phosphorylation in both the basal and induced state, with marked attenuation in the case of the Ala.sup(81) substitution. Replacing both Ser.sup(78) and Ser.sup(81) with Ala abolished nearly all phosphorylation of *CHOP* (Fig. 1D...

...The above results indicate that the stress-inducible phosphorylation of *CHOP* is dependent on two serines present in a context favorable for

phosphorylation by MAP k[redacted]ses (B9) (B10) . Members of th[redacted]family can be divided into...

...called Mpk2 and SAPK-2 (B13) (B14) . The last two groups respond to overlapping sets of stress signals, which include many that induce transcription of *CHOP*. p38 purified from stressed COS-1 cells phosphorylated bacterially expressed *CHOP* in vitro. Mutant *CHOP*, bearing the Ala.sup(78,81) substitution, was not a substrate for p38 (Fig. 2A). *CHOP* was not a substrate for SAPK-1 (beta) and was only weakly phosphorylated on Ser.sup(78) by ERK2. To explore further the relation between p38 and *CHOP*, we made use of a recently described, highly specific inhibitor of the enzyme, SB203580 (B15) . The inhibitor led to attenuation of the stress-inducible phosphorylation of *CHOP* on Ser.sup(78) and Ser.sup(81), whereas the activity of the related SAPK-1s was increased (Fig. 2, B and C). Collectively, these findings implicate p38 in the phosphorylation of *CHOP* in vivo. The ability of methyl methanesulfonate (MMS) to transcriptionally induce *CHOP* (B1) (B2) was not inhibited by SB203580. Thus, different stress-induced pathways regulate the transcription of *CHOP* and its phosphorylation...

...*CHOP* does not homodimerize. When present in cells, it forms stable heterodimers, predominantly with C/EBP (beta) (B3) . Indistinguishable quantities of C/EBP (beta) were found to be associated with wild-type and Ala.sup(78,81) *CHOP* in both stressed and unstressed cells (Fig. 3A). The DNA binding activity of *CHOP*, measured through use of a gel mobility-shift assay on a site that binds *CHOP*-C/EBP heterodimers, was also not affected by the Ala.sup(78,81) substitution (Fig. 3B). Phosphorylation therefore does not appear to alter the dimerization or DNA-binding properties of *CHOP*, although stress increases the ability of *CHOP* to activate transcription (B3) . Known *CHOP* target sequences also bind C/EBP dimers (B3) , and MAP kinases can phosphorylate the major dimerization partner of *CHOP*, C/EBP (beta) (B16) . Therefore, to evaluate possible effects of *CHOP* phosphorylation on its ability to activate transcription, we used a modified form of *CHOP* in which the leucine zipper was deleted and the protein fused to the DNA-binding domain of the yeast *transactivator* Gal4. Because such proteins lack a *CHOP* dimerization domain and bind DNA through the heterologous Gal4 peptide, the confounding effects of the stress-induced modification of the dimerization partners of *CHOP* is avoided. MMS treatment or overexpression of p38 markedly activated a reporter gene driven by Gal4 binding sites only when the *CHOP*-Gal4 chimeric protein was present (7.9 and 10 times as much, respectively); overexpression of SAPK-1 (beta) or ERK2 was without effect. The effect of MMS and p38 appeared to be dependent on *CHOP* phosphorylation because in cells expressing *CHOP*-Gal4 with an Ala.sup(78,81) substitution, reporter gene expression was not enhanced by these stimuli (Fig. 3C...

...Overexpression of *CHOP* leads to attenuated adipocytic differentiation of 3T3-L1 cells (B6) . This effect is dependent on the ability of the protein to dimerize and bind DNA. We compared the ability of wild-type and of Ala.sup(78,81) *CHOP* to inhibit adipocytic differentiation in 3T3-L1 cells. Both proteins inhibited differentiation; however, cells expressing wild-type *CHOP* showed less lipid accumulation than cells expressing the Ala.sup(78,81) substitution mutant (Fig. 3D). Thus, we conclude that Ser.sup(78) and Ser.sup(81) are necessary for the full biological activity of *CHOP*.

...

...Under favorable conditions, *CHOP* is not present in cells. Stress leads to accumulation of *CHOP* in the nucleus. Our results indicate that stress also leads to phosphorylation of the protein and that this modification results in enhanced transcriptional activation by *CHOP*. The *CHOP* accumulation in response to stress is apparently dependent on the activity of cellular kinases because it can be inhibited by the broad-spectrum kinase inhibitors 2-aminopurine and H7 (B1) . However, the signaling pathway that activates *CHOP* phosphorylation is distinct from the one that leads to *CHOP* expression; an inhibitor of p38 does not block *CHOP*

expression (Fig. 2B). Perhaps the potent ability of *CHOP* to modify cell growth and differentiation requires careful modulation beyond that provided by the mechanisms that control expression of the protein. The stress-activated p38 appears to serve a specific role in this fine-tuning of *CHOP* activity...

...Figure F1
Caption: Stress-induced phosphorylation of *CHOP* on Ser.sup(78) and Ser.sup(81). (A) Two isoforms of *CHOP* are present in stressed cells. NIH 3T3 cells were cultured in a medium with a low concentration of glucose [(Glu), 2 mM, 16 hours] or treated with tunicamycin (25 (mu) g/ml, 4 hours). *CHOP*, detected by protein immunoblotting with the 9C8 monoclonal antibody (B6) , migrates as a doublet with 11% SDS-polyacrylamide gel electrophoresis (SDS-PAGE). The form with less mobility is more acidic on isoelectric focusing (IEF). (B) Endogenous *CHOP* and Mycepitope-tagged *CHOP* [9E10 *CHOP* (B5)] from [.sup(32)P]orthophosphate-labeled NIH 3T3 cells (500 (mu) Ci/ml, 5 hours) that were treated with the stress-inducing alkylating agent MMS (100 (mu) g/ml, 3 hours) were immunoprecipitated with 9C8. Autoradiography (top) and protein immunoblotting with rabbit antiserum to *CHOP* (bottom) are shown. (C) Wild-type (WT) and mutant *CHOP*, immunoprecipitated from treated or untreated NIH 3T3 cells with the antibody to 9E10 (B17) , were digested with trypsin and the phosphopeptides separated by electrophoresis and...

...the tryptic cleavage sites, is shown above the autoradiograms (B19) . (D) Comparison of the in vivo phosphorylation of wild-type and Ala substitution mutants of *CHOP* from untreated cells and cells treated with MMS. Autoradiography (top) and *CHOP* immunoblot (bottom) are shown. (E) Schematic diagram of the *CHOP* protein. The region containing the stress-inducible phosphorylation sites is stippled, and the peptide sequence of this area, from four mammalian species, shows the conservation ...

...Figure Removed

Figure F2
Caption: Phosphorylation of *CHOP* by p38 MAP kinase. (A) Tryptic phosphopeptide maps of bacterially expressed *CHOP* phosphorylated in vitro with purified MAP kinases. Tagged forms of p38 (B13) , SAPK-1 (beta) (B20) , and ERK2 (B21) were expressed in COS-1 cells. Activated kinases, purified by the tag, were used to phosphorylate bacterially expressed wild-type or Ala.sup(78,81) *CHOP* with [(gamma) -.sup(32)P]ATP (B22) . The labeled proteins were evaluated ...SAPK-1 (beta) (B12) (B20) and myelin basic protein (MBP) for ERK2 (B21)]. (B) A p38-specific inhibitor, SB203580 (SB), blocks MMS-inducible phosphorylation of *CHOP*. NIH 3T3 cells expressing 9E10 *CHOP* were labeled with [.sup(32)P]orthophosphate as in Fig. 1B. The indicated concentration of SB203580 was added 30 min before MMS treatment. SB203580 inhibits MMS-induced phosphorylation of *CHOP* in a dose-dependent manner (top, autoradiogram) with no effect on 9E10 *CHOP* expression or on the induction by MMS of the endogenous *CHOP* (middle, *CHOP* protein immunoblot). In-gel kinase assay of SAPK-1s with GST-Jun as a substrate (B23) shows that the activity of SAPK-1s is increased (bottom). (C) Tryptic phosphopeptide mapping of *CHOP* from cells treated with MMS in the absence or presence of SB203580 (10 (mu) M) shows that inhibition of *CHOP* phosphorylation occurs at Ser.sup(78) and Ser.sup(81). The peptide marked "X" was constitutively phosphorylated...

...Figure Removed

Figure F3
Caption: Functional consequences of *CHOP* phosphorylation by p38. (A) Phosphorylation at Ser.sup(78) and Ser.sup(81) does not affect the dimerization of *CHOP* with C/EBP (beta) . Complexes of 9E10-tagged wild-type and Ala.sup(78,81) *CHOP* from NIH 3T3 cells were immunoprecipitated with 9E10 or nonimmune mouse immunoglobulin G (IgG) and immunoblotted with rabbit antisera to *CHOP* (top) or C/EBP (beta) (bottom). (B) Phosphorylation at Ser.sup(78) and Ser.sup(81) does not

affect the ability of *C[redacted]* to bind DNA. Nuclear extract[redacted]rom cells expressing wild-type or Ala.sup(78,81) 9E10 *CHOP* were used in a gel shift of a labeled *CHOP*-binding DNA fragment (B3) . Both proteins form indistinguishable complexes that were disrupted by antibodies to *CHOP* (3H8) and to the tag (9E10) but not by control IgG. (C) *CHOP* has a stress and p38-inducible *transactivation* domain. Wild-type or Ala.sup(78,81) *CHOP* was fused to the yeast Gal4 DNA-binding domain (B24) . NIH 3T3 cells were transfected with the indicated combinations of a reporter plasmid driven by two Gal4 binding sites (UASp59-luciferase, 5 (mu) g per plate), *CHOP*-Gal4 expression vectors (5 ng per plate), and kinase expression vector (5 ng per plate). Where indicated, the cells were treated with MMS (100 (mu...

...for luciferase assay. Shown are the mean and range values of a typical experiment performed in duplicate and reproduced four times. Wild-type and mutant *CHOP*-Gal4 were expressed in indistinguishable amounts in transfected cells. The inset shows a gel shift of a labeled Gal4 binding-site oligonucleotide by extracts from cells transfected with wild-type and mutant *CHOP*-Gal4 proteins. The arrow points to the complex supershifted with the 9C8 antibody to *CHOP*. (D) Ala.sup(78,81) *CHOP* is attenuated in its ability to inhibit adipocytic differentiation of 3T3-L1 cells. Pools of cells stably transfected with empty SRa retrovirus [PSI .sup(-) (B25)] or virus expressing either wild-type or mutant *CHOP* were induced to differentiate in vitro to adipocytes (B26) . The differentiation process was monitored by staining the fixed plates with the lipophilic dye Oil Red...

2/3,K/14 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136115942 CA: 136(8)115942c JOURNAL
Feedback inhibition of the unfolded protein response by GADD34-mediated dephosphorylation of eIF2.alpha.
AUTHOR(S): Novoa, Isabel; Zeng, Huiqing; Harding, Heather P.; Ron, David
LOCATION: Skirball Institute of Biomolecular Medicine, Department of Medicine, Kaplan Cancer Center, New York University School of Medicine, New York, NY, 10016, USA
JOURNAL: J. Cell Biol. DATE: 2001 VOLUME: 153 NUMBER: 5 PAGES: 1011-1021 CODEN: JCLBA3 ISSN: 0021-9525 LANGUAGE: English PUBLISHER: Rockefeller University Press

2/3,K/15 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133087544 CA: 133(7)87544s JOURNAL
Bile acid-induced activation of activator protein-1 requires both extracellular signal-regulated kinase and protein kinase C signaling
AUTHOR(S): Qiao, Dianhua; Chen, Weixing; Stratagoules, Elias D.; Martinez, Jesse D.
LOCATION: Arizona Cancer Center, Department of Radiation Oncology, University of Arizona, Tucson, AZ, 85724, USA
JOURNAL: J. Biol. Chem. DATE: 2000 VOLUME: 275 NUMBER: 20 PAGES: 15090-15098 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English PUBLISHER: American Society for Biochemistry and Molecular Biology

2/3,K/16 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130061837 CA: 130(6)61837c JOURNAL
Cloning of mammalian Ire1 reveals diversity in the ER stress responses
AUTHOR(S): Wang, Xiao-Zhong; Harding, Heather P.; Zhang, Yuhong;

Jolicoeur, Ethel M.; Kur[redacted], Masahiko; Ron, David
LOCATION: the Departments of Medicine, Cell Biology and the Kaplan Cancer Center, NYU Medical Center, Skirball Institute of Biomolecular Medicine, New York, NY, 10016, USA
JOURNAL: EMBO J. DATE: 1998 VOLUME: 17 NUMBER: 19 PAGES: 5708-5717
CODEN: EMJODG ISSN: 0261-4189 LANGUAGE: English PUBLISHER: Oxford University Press

2/3,K/17 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125138837 CA: 125(11)138837j JOURNAL
GADD153/CHOP, a DNA damage-inducible protein, reduced CAAT/enhancer binding protein activities and increased apoptosis in 32D cl3 myeloid cells
AUTHOR(S): Friedman, Alan D.
LOCATION: Div. Pediatric Oncol., Johns Hopkins Oncol. Cent., Baltimore, MD, 21287, USA
JOURNAL: Cancer Res. DATE: 1996 VOLUME: 56 NUMBER: 14 PAGES: 3250-3256 CODEN: CNREA8 ISSN: 0008-5472 LANGUAGE: English

2/3,K/18 (Item 1 from file: 35)
DIALOG(R)File 35:Dissertation Abs Online

01848873 ORDER NO: AADAA-I3024067
The role of C/EBPepsilon in terminal granulopoiesis
Author: Chih, Doris Yiafang
Degree: Ph.D.
Year: 2001
Corporate Source/Institution: University of California, Los Angeles (0031)
Source: VOLUME 62/08-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3484. 188 PAGES
ISBN: 0-493-35923-0

...interacting partners expressed in human leukocytes. The genes isolated fall into four large classes of factors: (1) other leucine zipper proteins such as CREB2, *CHOP*, C/EBPδ, and AF-17; (2) proteins involved in signal transduction, such as STAT6 and Sgn3; (3) protein inhibitor of activated STAT1...

...C/EBP&egr; may provide for the specificity of activation of myeloid-specific genes and heterodimerization with C/EBPδ or other factors may allow *transactivation* via DNA binding to specific sites (as in the case of lactoferrin promoter) or other mechanisms.

2/3,K/19 (Item 2 from file: 35)
DIALOG(R)File 35:Dissertation Abs Online

01629845 ORDER NO: AAD98-22927
A MODEL FOR NEUTROPHIL DIFFERENTIATION: SYNERGISTIC UPREGULATION OF THE G-CSFR PROMOTER BY RAS AND C/EBP(ALPHA) (C EBP$\ALPHA$)
Author: SMITH, LAURA TERESA
Degree: PH.D.
Year: 1998
Corporate Source/Institution: HARVARD UNIVERSITY (0084)
Source: VOLUME 59/01-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 93. 97 PAGES

...or deletion of these sites results in significant decreases in promoter activity. Co-transfection of both PU.1 and C/EBPα are able to *transactivate* either multimers containing the appropriate binding

site, or the native prom[redacted]r itself in cell lines that do[redacted]t express measurable levels of eith[redacted] of these...

...physically in GST-pull down assays via their DNA-binding domains, but since co-transfection of each of these factors does not result in additional *transactivation* above the levels seen with each factor alone, the significance of this interaction for the G-CSFR promoter is not clear. Co-transfection of the...
...EBPα site is necessary and sufficient for synergy and the region of C/EBPα necessary for synergy is area 3-5 in its *transactivation* domain. Furthermore, the synergy between Ras and C/EBPα also occurs with the M- and GM-CSF receptor promoters, suggesting that the CSF receptors...

...mechanism for the synergy between Ras and C/EBPα is not clear. Two proteins that attenuate Ras/C/EBPα synergy have been described, *CHOP* and Myb. *CHOP* is a C/EBP family member known to negatively regulate C/EBP function and Myb is an important hematopoietic transcription factor that activates the expression...
?